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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.
08/234,145	04/28/94	KUCHERLAPATI	400-0002-24

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18N2/0805

EXAMINER  
ZICHA, D

ART. UNIT	PAPER NUMBER
1004	

DATE MAILED: 08/05/97

Please find below and/or attached an Office communication concerning this application or proceeding.

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EXAMINER
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1804 23

DATE MAILED:

This is a communication from the examiner in charge of your application.  
COMMISSIONER OF PATENTS AND TRADEMARKS

#### OFFICE ACTION SUMMARY

- ☒ Responsive to communication(s) filed on 10/15/96
- ☐ This action is FINAL.
- ☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 D.C. 11; 453 O.G. 213.

A shortened statutory period for response to this action is set to expire three (3) month(s), or thirty days, whichever is longer, from the mailing date of this communication. Failure to respond within the period for response will cause the application to become abandoned. (35 U.S.C. § 133). Extensions of time may be obtained under the provisions of 37 CFR 1.136(a).

#### Disposition of Claims

- ☒ Claim(s) 4-7, 10-12, 1-3, 8, 9 is/are pending in the application.
- Of the above, claim(s) 1-3, 8, 9 is/are withdrawn from consideration.
- ☐ Claim(s) \_\_\_\_\_ is/are allowed.
- ☒ Claim(s) 4-7, 10-12 is/are rejected.
- ☐ Claim(s) \_\_\_\_\_ is/are objected to.
- ☐ Claim(s) \_\_\_\_\_ are subject to restriction or election requirement.

#### Application Papers

- ☐ See the attached Notice of Draftsperson's Patent Drawing Review, PTO-948.
- ☐ The drawing(s) filed on \_\_\_\_\_ is/are objected to by the Examiner.
- ☐ The proposed drawing correction, filed on \_\_\_\_\_ is ☐ approved ☐ disapproved.
- ☐ The specification is objected to by the Examiner.
- ☐ The oath or declaration is objected to by the Examiner.

#### Priority under 35 U.S.C. § 119

- ☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d).
- ☐ All ☐ Some\* ☐ None of the CERTIFIED copies of the priority documents have been
- ☐ received.
- ☐ received in Application No. (Series Code/Serial Number) \_\_\_\_\_
- ☐ received in this national stage application from the International Bureau (PCT Rule 17.2(a)).

\*Certified copies not received: \_\_\_\_\_

- ☐ Acknowledgment is made of a claim for domestic priority under 35 U.S.C. § 119(e).

#### Attachment(s)

- ☒ Notice of Reference Cited, PTO-892
- ☒ Information Disclosure Statement(s), PTO-1449, Paper No(s). \_\_\_\_\_
- ☐ Interview Summary, PTO-413
- ☒ Notice of Draftsperson's Patent Drawing Review, PTO-948
- ☐ Notice of Informal Patent Application, PTO-152

-SEE OFFICE ACTION ON THE FOLLOWING PAGES-

This application should be reviewed for errors.

Applicant's election with traverse of the IL8 antibody in Paper No. 21 is acknowledged. The traversal is on the ground(s) that applicants have traversed the restriction requirement on the grounds that the specification clearly groups individual antigens according to the type and function of the antigen and cites specification at page 22A, line 21 through page 22B. This is not found persuasive because the cited location lists a variety of polypeptides and proteins covering inflammation, autoimmunity and allergy. The cited conditions have different etiological bases, different manifestations and different outcomes and no one condition can be controlled or managed by treatment with one particular antibody to one particular antigen or protein. Contrary to further arguments, an antibody binds specifically to its cognate antigen and not to a "set of antigens" related by function. The only function seen to be in common of antibodies generated to the listed proteins/antigens is the fact that the antibody would bind to its cognate antigen.

The requirement is still deemed proper and is therefore made FINAL.

Claims 4-7 and 10-12, with the IL8 species elected, are active and examined in this Office Action.

The 5,591,669 patent to Krimpenfort ('669 patent) is considered to be enabling for heavy chain J region knockout, is granted the filing date of the grandparent application as its priority date (December 5, 1988) and therefore qualifies as proper prior art.

Claims 4-7, 10 and 12 are rejected under 35 U.S.C. 112, second paragraph, as failing to set forth the subject matter which applicant(s) regard as their invention. Claims 7 and 12 are

grammatically incorrect. Claims 7 and 12 appear to be claiming as tumor antigens human IgE, HIV-1 envelope glycoprotein, 5 different viruses, etc. Clarification is required. Regarding claim 10, the phrase "other than human glycosylation" is vague and unclear since human glycosylation is not defined in the specification and "other than human glycosylation" lacks metes and bounds. Does a simple change of a single oligosaccharide or the lack of a single oligosaccharide moiety qualify as "other than human glycosylation" if all the other oligosaccharide moieties are the same?

Regarding claim 4, the word "xenogeneic" only has meaning when compared to a different species. The claimed immunoglobulin is not different from the immunoglobulin produced by the host species. The meaning of "xenogeneic" in this context is vague and unclear when the immunoglobulin is considered alone. In product by process claims, the product is examined and the claimed immunoglobulin can be produced by chemical peptide synthesis methods, in vitro or in vivo and then purified, lacking evidence to the contrary.

Claims 4-7 and 10-12 are rejected under 35 U.S.C. 112, first paragraph, as containing subject matter which was not described in the specification in such a way as to enable one skilled in the art to which it pertains, or with which it is most nearly connected, to make and/or use the invention. The instant specification discloses transgenic mice incapable of producing endogenous murine immunoglobulins. The specification further discloses that the mice contained integrated human DNA from the human immunoglobulin loci and that the mice were produced by spheroplast fusion of YACs with ES cells. However, the specification prophetically discloses that the mice containing the human immunoglobulin DNA segments could be mated to the mice having inactivated endogenous immunoglobulin loci at page 73, top

paragraph, of the specification. The next example discloses that mice containing the human immunoglobulin gene segments were immunized. It is not clear from the specification that the cross-breeding scheme prophetically disclosed was successful and that human antibodies were obtained from mice having endogenous immunoglobulin loci inactivated. The description of the splenocyte fusion and testing of antibody production by the hybridomas does not preclude production of fully human antibodies by hybridomas obtained from mice still producing murine immunoglobulins.

Regarding claims 4 and 5, the specification is not enabling for the production of human antibodies as claimed since the specification fails to disclose that cross bred mice were successfully obtained, since the specification fails to disclose the method of detection of human antibodies and evidence that the antibodies produced to injected human IgE produced were human antibodies. Regarding claims 6 and 7, the specification fails to disclose binding assays showing the immunospecificity of the expressed antibody for human IgE. Immunospecificity requires a showing that the antibody reacts specifically with the immunizing antigen and does not cross-react with other antigens.

Regarding claims 10-12, the specification fails to disclose methods of determining glycosylation differences on the produced antibody. There is no teaching in the specification that human glycosylation is different than mouse glycosylation either in general in B cells or specifically with respect to the claimed antibody. The specification fails to disclose methods of determining glycosylation differences or that one of skill would be able to differentiate mouse antibodies from human antibodies based on glycosylation differences. Further, the differences between human glycosylation and "other than human glycosylation" are not disclosed, and no guidance is provided in the

specification for determining when glycosylation is "other than human".

Regarding the claims, the specification fails to disclose that the human immunoglobulin genes inserted via YAC fusion are capable of undergoing rearrangement to produce a functional variable region; that once a functional variable region was produced that the human mu constant region was capable of being functionally linked to the produced V region or capable of undergoing isotype switching so as to be functionally linked to other constant regions. The prior art cited below (Bruggeman) obtained variable, D and J segment rearrangement using other methods. The instant specification is denied the priority dates of the parent applications for transgenes capable of undergoing isotype switching since the specification does not disclose or describe either methods for making the transgene or the transgenes which would have that capability. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make the invention commensurate in scope with these claims. The specification, while being enabling for HPRT, does not reasonably provide enablement for a xenogeneic DNA wherein the DNA is a human immunoglobulin gene. This rejection is necessitated by the issuance of the patents to Lonberg containing the Cox declaration which states that the human immunoglobulin DNA locus as described in the instant specification was not, and could not, be cloned at the time the claimed invention was made, and secondly, in view of references with significantly later postfiling publication dates specifically stating that isotype switching to downstream isotypes had not yet been achieved using the described human Ig loci contained in YACs.

The Cox declaration states that there were no reports of the cloning in YAC vectors of the region spanning the human delta and gamma3 genes. This region is not readily or predictably cloned

and the problem, as explained by the declarant, is possibly due to instability of that region of the human genome in certain cloning vectors, such as YACs. Thus, the art had not reported the cloning of an intact human IgH locus containing the V, D, J, mu, delta and gamma sequences, let alone an intact human IgH locus cloned in a YAC.

The published art was not aware of how to make a transgenic animal containing a transgene capable of undergoing isotype switching to produce human isotypes downstream of IgM as evidenced by Taki et al (1993), disclosing that the authors did not know how to make a transgene that could undergo cis-isotype switching where endogenous sequences were not involved. Morrison, writing a review of the field in Nature (1994), discussed the teachings of Green et al., which referenced two Kucherlapati published PCT applications, (page 812, column 2, bottom):

"In Green and colleague's mice, only the mu heavy chain contributes significantly to the circulating antibody population, and these mice are unable to undergo the isotype switching characteristic of the mature antibody response. But in the mice of Lonberg et al., the heavy-chain locus contains both a mu and gamma heavy chain with switch sites, and these authors show that class switching occurs. So they have reconstituted the essential part of a human-antibody-producing response in a mouse and the system could be used both to study control of antibody production as well as to produce specific human antibodies".

In view of the foregoing, the instant specification is not enabling for the human IgH locus containing the V, D, J, mu, delta and gamma sequences cloned into a YAC capable of undergoing isotype switching, nor for a transgenic animal containing the IgH locus-YAC.

The specification of the instant application regarding the actual production of transgenic mice containing human immunoglobulin genes produced by fusion of spheroplasts containing YACs with ES cells is speculative and does not present any working examples showing actual cloning of human immunoglobulins (monoclonals) and that if such mice were actually

obtained, that human antibodies would be expressed from the human IgH locus. In view of the lack of actual working examples and evidence presented showing that the DNA containing the human immunoglobulin locus could not be obtained by applicant's method at the time the claimed invention was made, the specification is not enabling for the method, or animals produced by the method, as claimed.

The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

A person shall be entitled to a patent unless --

(e) the invention was described in a patent granted on an application for patent by another filed in the United States before the invention thereof by the applicant for patent, or on an international application by another who has fulfilled the requirements of paragraphs (1), (2), and (4) of section 371(c) of this title before the invention thereof by the applicant for patent.

Claims 4, 5 and 10 are rejected under 35 U.S.C. 102(e) as being anticipated by Lonberg. Lonberg discloses xenogeneic immunoglobulins which are human; see Column 88. Therefore, the reference anticipates the claims. This rejection addresss the product only.

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

(a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

This application currently names joint inventors. In considering patentability of the claims under 35 U.S.C. 103(a), the examiner presumes that the subject matter of the various



claims was commonly owned at the time any inventions covered therein were made absent any evidence to the contrary. Applicant is advised of the obligation under 37 CFR 1.56 to point out the inventor and invention dates of each claim that was not commonly owned at the time a later invention was made in order for the examiner to consider the applicability of 35 U.S.C. 103(c) and potential 35 U.S.C. 102(f) or (g) prior art under 35 U.S.C. 103(a).

Claims 4-6, 10 and 11 are rejected under 35 U.S.C. 103(a) as being unpatentable over Krimpenfort (USPN 5,5591,669) taken with Lonberg (USPN 5,545,806), Bruggemann (PNAS, 1989) and Surani (USPN 5,545,807). This rejection addresses the product by process claims wherein the process of producing the product is considered. Krimpenfort discloses and claims transgenic mice having a deletion of nucleotides from the cognate endogenous immunoglobulin heavy chain allele wherein said deletion comprises a deletion of J segments of the variable region and therefore discloses disruption of an endogenous heavy chain allele. Inhibition of light chain expression would have been obvious in view of the known heavy chain-light chain structure of complete immunoglobulin molecules and in view of the stated intention by Krimpenfort to inhibit immunoglobulin production. It was well known at the time the claimed invention was made that a functional immunoglobulin consisted of heavy chains and light chains. B cells lacking functional genes for either the light chain or the heavy chain would have been incapable of producing functional immunoglobulins since a functional immunoglobulin required expression of both chains. In column 10, lines 21-30, Krimpenfort discloses targeted insertion of a transgene into the TCR alpha chain constant region, analogous to the light chain of immunoglobulins, in order to disrupt expression of the alpha chain. Krimpenfort thus discloses inactivation of the TCR since expression of both the heavy chains and light chains is necessary

to obtain a functional receptor. Therefore inactivating expression of the immunoglobulin light chain would have been obvious over the inactivation of the immunoglobulin heavy chain, lacking evidence to the contrary, since inactivation of either chain would have resulted in inhibition of immunoglobulin expression and Krimpenfort discloses inhibiting the expression of an analogous protein, the TCR, by inactivating expression of either the heavy chain (beta) or the light chain(alpha).

One of ordinary skill would have had a reasonable expectation of success in achieving inactivation of the kappa J region using the methods of Krimpenfort in view of the teachings of Krimpenfort disclosing that the heavy and light chains of the immunoglobulins are well characterized (column 12, lines 54-56). Krimpenfort discloses the structural analogy between the heavy and light chains of the T cell receptor and the heavy and light chains of the immunoglobulins and discloses that modifications made at the nucleotide level to the genes encoding the polypeptide chains of the TCR may be made in a similar manner at the nucleotide level of the immunoglobulin genes as well. Krimpenfort explicitly teaches the addition of inserted transgene that may be capable of either facilitating or inhibiting the maturation of a lymphatic cell type (column 6, lines 55-65) and further that the transgene may be from any species and therefore xenogeneic. **Krimpenfort differs from the claims in that the reference fails to disclose production of xenogeneic antibodies.** However, the secondary references, Bruggemann, Lonberg (USPN 5,545,807) and Surani (USPN 5,545,806), cure the deficiency. Bruggemann discloses a heavy chain transgene containing genes encoding the human and mouse mu constant regions. Bruggemann discloses that xenogeneic antibodies were produced from a transgene containing both mouse and human mu constant regions and therefore teaches an animal comprising detectable xenogeneic antibodies in its sera since the serum titer of transgenic IgM

was about 50 ug of antibody/ml. Bruggemann further discloses that 4 transgenic hybrids express mouse and human u chains in the serum (page 6711, column 1, last paragraph). Surani discloses that the transgenic mice can produce xenogeneic antigen specific antibodies (column 6, example 3). Lonberg discloses human antibodies having human IgG heavy chains and human light chains, and discloses such production in a knockout background, thus disclosing xenogeneic antibodies. Lonberg is cited to disclose transgenes encoding both human IgM, human IgG and human light chains, the heavy chain transgenes being capable of undergoing isotype switching, the isolation of B cells and the production of monoclonal antibodies to a specified human antigen.

The motivation to combine the references can be found within each reference. It would have been obvious to one of ordinary skill to modify the mouse of Krimpenfort by adding a transgene encoding a functional immunoglobulin molecule in view of the teachings of Krimpenfort that the inserted transgene may be capable of either facilitating or inhibiting the maturation of a lymphatic cell type (column 6, lines 55-65) and further that the transgene may be from any species and therefore xenogeneic. It would have been obvious to one of ordinary skill to knockout endogenous immunoglobulin production, as taught by Krimpenfort, using targeting constructs directed to the JH region to inactivate both endogenous heavy chain and light chain alleles, and to further modify the mouse genome by the addition of transgenes capable of facilitating maturation of a lymphatic cell type. IgM is known in the art to be required for lymphocytic cell maturation.

Bruggemann provides further motivation to produce a transgenic mouse comprising human xenogeneic immunoglobulins in a knockout background on page 6712, column 1, last paragraph, wherein it is stated "It would be useful to have transgenic mice that have nonfunctional endogenous immunoglobulin loci so that

they can only make human antibodies". Thus, it would have been obvious to one of ordinary skill to add a transgene encoding xenogeneic immunoglobulin in order to obtain a transgenic mouse in which the endogenous immunoglobulin genes have been inactivated in order to obtain a mouse capable of producing human immunoglobulins. Bruggemann therefore also provides the motivation to add other transgenes, encoding for example, human IgG, since the repertoire of human antibodies encompasses IgM, IgD, IgG, IgA and IgE.

Regarding claims 4-6, and 10, Surani discloses transgenic mice capable of making an immune response to the SRBC antigen. It would have been obvious to one of ordinary skill to immunize the mice with other antigens, such as KLH-DNP, in view of the teachings of Bruggemann suggesting that mice containing the human antibody genes and expressing human antibodies would not be tolerant to most human determinants and could be used to make human antibodies against human antigens (page 6712, column 1, top paragraph), and further in view of Surani, disclosing production of IgM antibodies containing a human heavy chain against SRBC antigens. Surani provides the reasonable expectation of success of producing antibodies against particular antigens since Surani used the same transgene disclosed by Bruggemann. Lonberg discloses production of xenogeneic human immunoglobulins reactive with a preselected antigen. Regarding claim 10, the glycosylation of the produced protein would be expected to be that of the host system, lacking evidence to the contrary.

Regarding claim 11, it would have been obvious to one of ordinary skill to produce monoclonal antibodies using B cell obtained from the mice containing the transgenes encoding xenogeneic antibodies since monoclonal antibody production was old and well known in the art at the time the claimed invention was made. Lonberg discloses the production of hybridomas from B cells containing human antibody genes in column 2.

Accordingly, the modification of the xenogeneic immunoglobulin taught by Krimpenfort by adding a transgene encoding a human mu immunoglobulin heavy chain as suggested by Bruggeman and Surani, or both a human IgG heavy chain and a human light chain as suggested by Lonberg, in order to produce a xenogeneic immunoglobulin was within the ordinary skill in the art at the time the claimed invention was made. From the teachings of the references, it is apparent that one of ordinary skill would have had a reasonable expectation of success in producing the claimed invention, and therefore, the invention as a whole is prima facie obvious.

Claim 11 is rejected under 35 U.S.C. 103(a) as being unpatentable over Lonberg as applied to claims 4, 5 and 10 above, and further in view of Lonberg. Claims 4, 5 and 10 are rejected for reasons as set forth above. Lonberg clearly discloses the desirability of producing monoclonal antibodies in column 2 and elsewhere throughout the specification. Therefore, it would have been obvious to one of ordinary skill to follow the methods set forth in Lonberg to immortalize B cells produced in the transgenic mouse, produce hybridomas and collect the human antibodies. From the teachings of the references, it is apparent that one of ordinary skill would have had a reasonable expectation of success in producing the claimed invention, and therefore, the invention as a whole is prima facie obvious.

Claims 7 and 12 are rejected under 35 U.S.C. 103(a) as being unpatentable over Krimpenfort, Lonberg, Bruggemann and Surani as applied to claims 4, 5, 6, 10, and 11 above, and further in view of James et al. and Ko et al. Claims 4-6, 10 and 11 were rejected for reasons as set forth above. Ko teaches the availability of human recombinant IL8 and therefore implicitly discloses that human IL8 protein has been obtained and that both the amino acid sequence and nucleotide sequence are known. Ko further discloses use of human recombinant IL8 as an immunogen in mice for the

purpose of raising murine antibodies to human IL8. Ko additionally discloses the production of monoclonal antibodies to human IL8 (page 230, column 1). It would have been obvious to one of ordinary skill to immunize the mouse of Krimpenfort, Lonberg, Bruggemann and Surani with human IL8 in order to produce human sequence immunoglobulins against the human IL8 antigen in view of the stated desire of Lonberg to produce human immunoglobulins against human immunogens; in view of the stated desire of Bruggemann to produce human immunoglobulin in mice incapable of producing endogenous immunoglobulins; and in view of the teachings of James disclosing the desirability to produce human antibodies against human interleukins which might be used to reverse graft rejection and treat autoimmune disease (page 25, column 2, second full paragraph). Although James does not specifically disclose production of antibodies to IL8, Ko discloses that IL8 is a cytokine involved in inflammatory reactions, known to accompany autoimmune disease. One of ordinary skill would have had a reasonable expectation of success in producing human immunoglobulins to human IL8 in view of the success obtained by Lonberg, showing antibodies containing human heavy chains reactive to human antigens (CEA) in an endogenous immunoglobulin knock-out background.

The modification of the xenogeneic immunoglobulin by substituting the human IL8 antigen used for immunogenic stimulation was within the ordinary skill in the art at the time the claimed invention was made. From the teachings of the references, it is apparent that one of ordinary skill would have had a reasonable expectation of success in producing the claimed invention, and therefore, the invention as a whole is prima facie obvious.

Claims 7 and 12 are rejected under 35 U.S.C. 103(a) as being unpatentable over Lonberg as applied to claims 4-6, 10 and 11 above, and further in view of James et al. and Ko et al. Claims

4-6, 10 and 11 were rejected for reasons as set forth above. Ko teaches the availability of human recombinant IL8 and therefore implicitly discloses that human IL8 protein has been obtained and that both the amino acid sequence and nucleotide sequence are known. Ko further discloses use of human recombinant IL8 as an immunogen in mice for the purpose of raising murine antibodies to human IL8. Ko additionally discloses the production of murine monoclonal antibodies to human IL8 (page 230, column 1). It would have been obvious to one of ordinary skill to immunize the mouse of Lonberg with human IL8 in order to produce human sequence immunoglobulins against the human IL8 antigen in view of the stated desire of Lonberg to produce human immunoglobulins against human immunogens and in view of the teachings of James disclosing the desirability to produce human antibodies against human interleukins which might be used to reverse graft rejection and treat autoimmune disease (page 25, column 2, second full paragraph). Although James does not specifically disclose production of antibodies to IL8, Ko discloses that IL8 is a cytokine involved in inflammatory reactions, known to accompany autoimmune disease. One of ordinary skill would have had a reasonable expectation of success in producing human immunoglobulins to human IL8 in view of the success obtained by Lonberg, showing antibodies containing human heavy chains reactive to human antigens (CEA) in an endogenous immunoglobulin knock-out background.

The modification of the xenogeneic immunoglobulin of Lonberg by substituting the human IL8 antigen used for immunogenic stimulation as suggested by Ko and James was within the ordinary skill in the art at the time the claimed invention was made. From the teachings of the references, it is apparent that one of ordinary skill would have had a reasonable expectation of success in producing the claimed invention, and therefore, the invention as a whole is prima facie obvious.

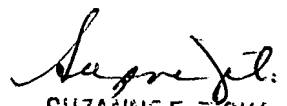
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No claim is allowed.

Papers related to this application may be submitted to Group 1800 by facsimile transmission. Papers should be faxed to Group 1800 via the PTO FAX center located in Crystal Mall 1. The faxing of such papers must conform with the notice published in the Official Gazette, 1096 OG (30 November 15, 1989). The CM1 official Fax Center number is (703) 305-3014 or (703) 305-4242.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Examiner Suzanne Ziska, Ph.D., whose telephone number is (703)308-1217. In the event the examiner is not available, the examiner's supervisor, Jasemine Chambers, Ph.D., may be contacted at phone number (703) 308-3153.

  
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